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Case Reports

AN UNUSUAL CASE OF TUBERCULOUS MENINGITIS

A. R. ARMSTRONG, M.D., B.Sc.,*
Hamilton, Ont.

THIS IS A REPORT of an autopsy performed on a case of tuberculous meningitis, which is instructive from two points of view. It illustrates the development of meningitis from a tuberculous cord lesion and also the value and limitations of frontal burr holes in the treatment of meningitis.

During the three years since the demise of the patient, the treatment of tuberculous meningitis has undergone a complete change. Today oral administration of isoniazid with a cover of intramuscular streptomycin is employed. Intrathecal administrations, as a rule, are no longer in use. Mention is made, however, of the therapy employed in this instance, because it is necessary to have this information to interpret fully the various signs and symptoms described, some of which were due to the treatment.

CLINICAL DATA

One year before his death a 15-year-old boy had visited a cousin who later was found to be an open case of tuberculosis. During this visit, which lasted a week, he had slept in the same bed as the cousin. About the same time he had received a severe contusion to his upper thoracic spine during a hockey game. Eight months before death he developed a cold, headache, backache, nausea and vomiting. Successive treatments with penicillin, streptomycin, and aureomycin had failed after several weeks to improve his condition. He was admitted to the Hospital for Sick Children, Toronto, with the following findings: He was not acutely ill. He had pain in the posterior neck muscles. There was slight limitation of neck flexion. The superficial lymph nodes were slightly enlarged. Kernig's sign was negative. There was no tache cerebrale. White cell count 8,000 with 81% neutrophils, 10% monocytes, and 8% lymphocytes. A tuberculin test was positive to 1/10 mgm. O.T. A chest radiograph was normal.

After three days' treatment with aureomycin he was noted to have become increasingly drowsy. A lumbar puncture revealed a yellow fluid with a pressure of 210 mm. water, cell count of 450 (all lymphocytes), protein of 1086 mgm. %, sugar of 45 mgm. % and chlorides of 648 mgm. %. Culture and guinea-pig inoculation of the fluid failed to demonstrate tubercle bacilli. Because of a tentative diagnosis of tuberculous meningitis, he was given 0.5 gm. streptomycin daily intramuscularly. Because of the Froin syndrome, frontal burr holes were made and 25,000 units of streptomycin were given daily, alternately into each lateral ventricle.

The boy did well at first but after about three weeks he developed fever, stupor, and vomiting. His abdominal reflexes were absent. There were bilateral upgoing toes and bilateral ankle clonus. The intraventricular drug was stopped. After about three weeks, by which time he was considerably improved, it was re-started but given only every third day and for a three-week period. The daily intramuscular streptomycin, on the other hand, was not stopped and, in fact, was continued for some five months.

Slight weakness of the lower extremities was now observed. This gradually increased and he also began to develop urinary retention with overflow. He had aching pains in the neck and back which were made worse by turning his head from side to side.

Three and one-half months before death examination revealed a sensory loss, diminished temperature discrimination, diminished vibration sense, and reduced touch appreciation from the 9th thoracic segment down. There was marked symmetrical leg weakness. The abdominal reflexes were absent. The leg reflexes were active. There were bilateral upgoing toes and bilateral ankle clonus. There was urinary retention with overflow.

Radiography after cisternal Pantopaque injection demonstrated an apparently complete block at the 4th thoracic vertebra. A laminectomy involving the spines of the 1st, 2nd, and 3rd thoracic vertebræ was performed, and at operation the cord was noted to be swollen and felt cystic, but attempts at aspiration failed. A few drops of yellow pus were expressed through an opening in the arachnoid, but no tubercles were seen.

He was now treated with intraventricular streptomycin plus progressively increasing amounts of old tuberculin. These at first resulted in brief blackouts and finally in complete unconsciousness, slowed pulse, depressed irregular respirations, and upturned eyes directed to the left. The tuberculin was henceforth omitted and he rallied well. Examination just before transfer to the Mountain Sanatorium, and one month before death, elicited stiffness and soreness of his neck (flexion 20%) and revealed papilloedema and secondary optic atrophy. Ventricular punctures showed pressures of 40-60 mm. water on the right and 90-140 on the left. There were no cells. Other significant findings were sensory loss below the 4th thoracic segment, urinary retention, decubitus ulcerations of large extent, absent abdominal reflexes, and accentuated knee and ankle jerks. Plantar reflexes and ankle clonus were absent. A radiograph showed that the Pantopaque had now descended as far as the 11th thoracic vertebra.

The record after admission to the Mountain Sanatorium shows that he was acutely ill with a fever of 102° F. and a slow pulse. He had severe recurrent headaches and was mentally disorientated. He had transient blackouts during which his eyes deviated to the left and his right pupil was larger than the left. Pulsating tumours were seen in the areas of the burr holes, but on intraventricular puncture no increased pressure was demonstrated. The fluid was clear and colourless, and no pellicle formed on letting it stand. The sugar was 73 mgm. %; the chlorides 706 mgm. %. No tubercle bacilli were obtained on culture.

The intraventricular puncture did not improve the patient and, indeed, his blackout spells became increased. He died during one of these attacks.

AUTOPSY FINDINGS

The abnormal autopsy findings were almost completely limited to the brain and spinal cord.

On reflecting the scalp, meninges and brain were seen bulging through the burr holes, this being more marked on the left side. After removing the skull cap this bulging was very noticeable (Fig. 1). The subarachnoid space was dry above the tentorium. The subarachnoid space below the tentorium was bulging with a large

*From the Laboratories of the Mountain Sanatorium.
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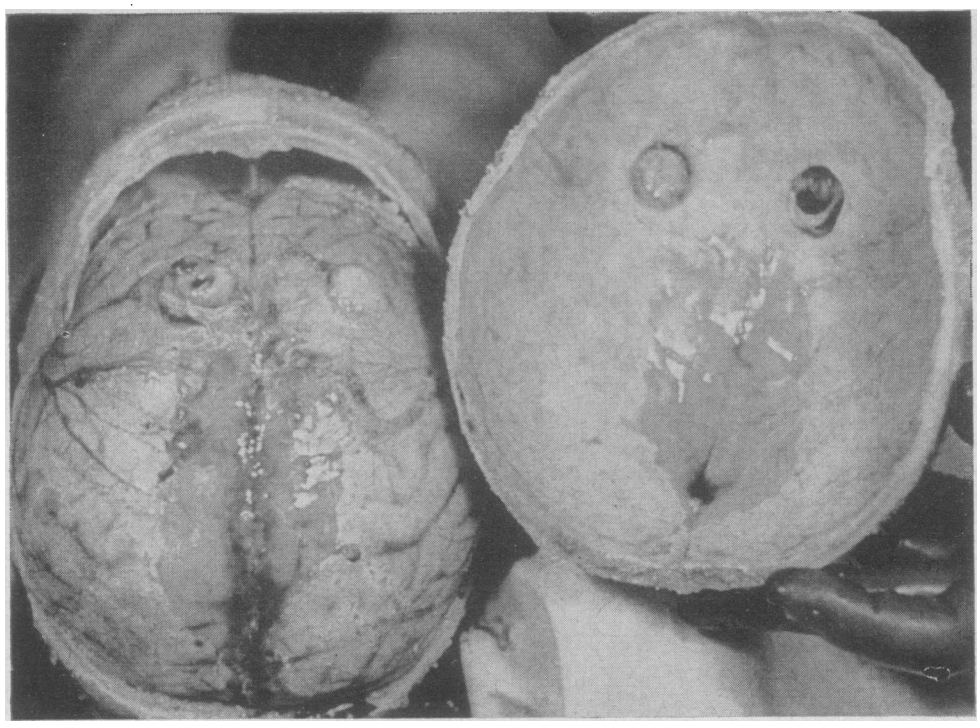

Fig. 1.—The skull cap has been removed. Burr holes are seen in the under-surface of the cap. Brain and meninges are bulging upwards in the region of the burr holes, this bulging being more marked on the left side.

amount of clear fluid as seen on cutting the tentorium. Puncture of the arachnoid below the tentorium permitted an amount of fluid somewhat in excess of 100 c.c. to gush up into the calvarium. All ventricular cavities, the aqueduct, and the foramina of the fourth ventricle showed a marked dilatation. The right lateral ventricle was filled with blood clot. This clot is shown in Fig. 2. The lateral wall of the anterior horn contained a 2.5 x 1 cm. area of softening which extended for a depth of 0.5 cm. laterally. Some recent needle-puncture wounds were visible in the white matter on the left side in the region of the anterior horn and one of these can be made out in the same photograph.

The only gross evidence of tuberculous meningitis was the presence of some adhesions around the cerebral peduncles and pons.

When the spinal cord was exposed by laminectomy, the cord substance was found herniating backwards through the dura and into a fibrous pouch in the region of the spines of the 2nd and 3rd thoracic vertebræ (the operative laminectomy site). The cord substance was softened and an almost complete transverse myelectomy had occurred. The softened herniating cord is seen in Fig. 3. Exudate and adhesions in the subarachnoid space below this area were sufficiently dense to have obstructed the passage of spinal fluid in a downward direction. Exudate and adhesions were also found between the dura and arachnoid. These exudates and adhesions are just visible in the photograph.

A microscopical search for tuberculosis showed that at one point in the leptomeninges covering the lower part of the cord there was a small but typical tubercle with caseation and acid-fast bacilli. A low-power photomicrograph illustrates this caseous focus (Fig. 4). Elsewhere there

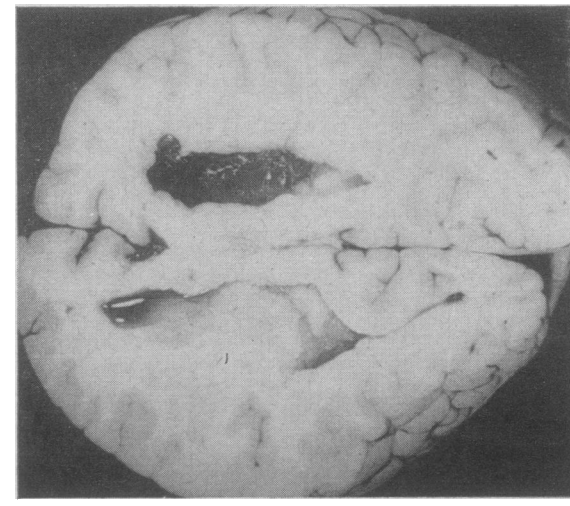

Fig. 2.—The lateral ventricles have been exposed by horizontal section. A large blood clot is seen in the right lateral ventricle. A needle puncture wound can be seen in the white substance of the brain just anterior to the left lateral ventricle.

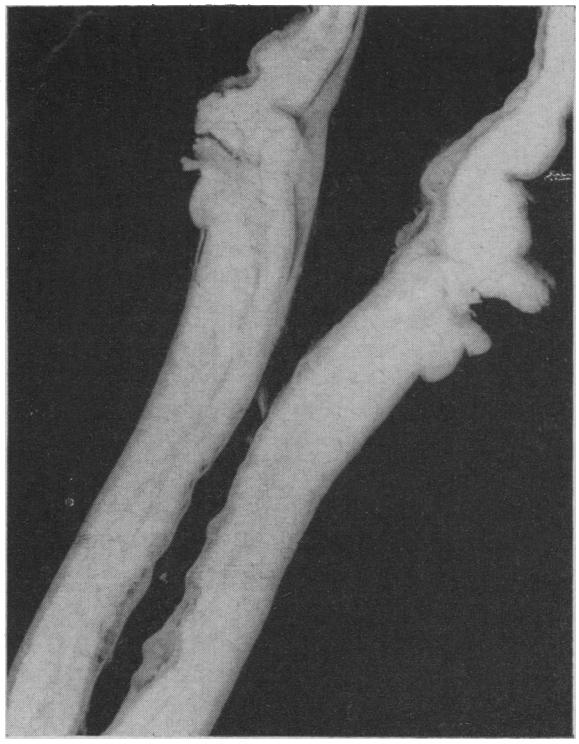

Fig. 3

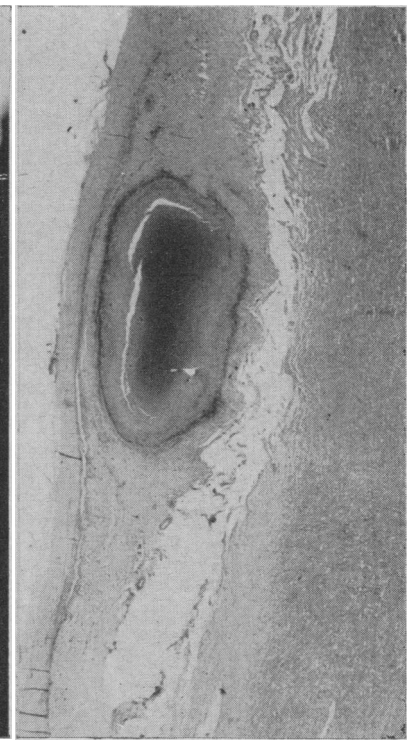

Fig. 4

Fig. 3.—Cervical and first part of the thoracic spinal cord after sagittal section directly through the posterior to the anterior surface, the two halves being spread apart like the leaves of a book. The bulging, softened cord is projecting posteriorly. Adhesions, subdural and subarachnoid exudate can be made out below this area. **Fig. 4.**—Low-power photomicrograph of a longitudinal section of leptomeninges and lower spinal cord showing an oval area of caseation in the subarachnoid space.

were only adhesions and lymphocytic infiltrations. This one active lesion was the only proof of tuberculosis found anywhere in this body. The cord showed extensive degeneration at the laminectomy site. The microscopical sections of the cerebral meninges showed only fibrosis, scattered diffuse lymphocytic infiltrations, and occasional small groups of macrophages. One or two rounded areas of fibrosis suggested healed tubercles. The lungs were entirely normal.

Associated with the cord lesion as a result of his paraplegia were extensive decubitus ulcerations, the largest measuring 9 cm. in diameter.

DISCUSSION

The probable sequence of events in this case was as follows. A small primary tuberculous lesion, which subsequently completely healed, was contracted perhaps in the lungs, perhaps in the tonsils or intestine, as a result of the earlier contact with the tuberculous relative. Hæmatogenous spread preceded the healing of this primary focus and originated a small caseous spinal tuberculous focus. The role, if any, of the trauma to the spine in determining the localization of this new lesion might have been in disturbing the blood supply. It is conjectured that the small caseous lesion ruptured spontaneously into the subarachnoid space, setting up the tuberculous leptomeningitis and cerebral meningitis. A fibrosis of the membranes in the region of the 4th thoracic vertebra then produced Froin's syndrome.

The cerebral meningitis was promptly rendered inactive by streptomycin and old tuberculin, but enlargement of the cord lesion became manifest clinically by transverse myelitis. At operation it may be presumed that tuberculous material entered the subdural space and that a pachymeningitis then arose.

Contraction of the scar tissue, resulting from the healing cranial meningitis, soon produced a complete block at the level of the tentorium, giving rise to an internal and partial external hydrocephalus. Increased pressure caused herniation of the cord at the operation site. Although a careful search for remains of the original disease at this point was made, none was found. It is thought that the lesion was in part expressed at operation and it is likely that the remainder healed as a result of the extensive antibiotic treatment.

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SUMMARY

A case of tuberculous meningitis, believed to have developed from rupture of a caseous thoracic cord lesion (which was producing a transverse myelitis) into the subarachnoid space, is reported. The cord lesion developed following a back injury and subsequent to known exposure to tuberculosis. Although the cerebral tuberculous meningitis was inactivated by treatment, inflammatory adhesions around the upper brain stem had produced obstruction of the subarachnoid space. The increased pressure below the tentorium cerebelli was responsible for the partial herniation of the spinal cord through the site of laminectomy. A healing tuberculous leptomeningitis was present throughout the whole length of the cord, but only a single active lesion was found. Acid-fast bacilli were readily demonstrable in this caseous subarachnoid focus.

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ROOT PAIN FROM CERVICAL OSTEOARTHRITIS SIMULATING ANGINA PECTORIS

H. R. L. DAVIS, M.D., *Vancouver, B.C.*

THE HEART seems to have pre-empted the precordial region as a common site for pain. Any layman can tell you that if there is also pain running down the left arm, it is angina. This anginal pain is usually helped by the nitrites.

A 71-year-old professional man had had myocardial enlargement for many years, with left bundle branch block and shortness of breath. He had been granted a disability pension because of myocardial degeneration, and also had entitlement for arthritis. In 1946 the pain in his left arm was so severe that he was obliged to give up work. Among others, he consulted physicians at the Montreal Neurological Institute, where, I understand, a diagnosis of osteoarthritis and disc syndrome was made. He reacted well to halter traction.

In the following years the patient gradually developed anginal pain, which reacted to nitrites. More severe pain also occurred in the precordial region; it was thought to be cardiac and was so severe as to require morphine, although it was at a lower level than in 1946. In December 1953, the pains became very severe. There were as many as ten attacks a day, and they were not relieved by nitrites. The patient continued to suffer, and by May 1954 the situation was very difficult.

Although, in view of the established diagnosis of heart disease, it had seemed logical to assume that the pain, which occurred in the usual site of angina pectoris, was cardiac in origin, it occurred to me that something might have been missed. There seemed no reason to doubt that the patient had heart disease, but might not the severe pain be due to something else?

The case was reviewed. There was still atrophy of the intrinsic muscles of the left hand, which had been present in 1946. Skin sensation to sharp point showed hyperæsthesia at the level bilaterally of the third thoracic nerve. A radiograph of the cervicodorsal spine revealed lipping. This seemed sufficient to establish a diagnosis of root pain due to arthritis, a condition which it was thought could be helped by halter traction or by deep x-ray treatment.

As the patient had entitlement for both arthritis and heart disease, I referred him to Shaughnessy Hospital for halter traction. Traction was begun at 15 pounds twice a week. In the second week, when the patient was receiving a pull of 22 pounds, there was a sudden feeling of relief. This has continued during the past two months (to July 16, 1954). In all, the patient received about ten treatments with a maximum pull of 27 pounds. During a recent interview the patient stated that he now has only mild discomfort in the precordial region and this reacts well to nitroglycerin.

There are perhaps similar cases which would benefit from study. Many writers have drawn attention to similar pains at various levels of the spine, although I do not remember having seen or read of a case similar to this.

736 Grenville St.